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Lead poisoning in cattle associated with batteries recycling: High lead levels in milk of nonsymptomatic exposed cattle

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Abstract

Lead poisoning associated to residuals of batteries recycling was diagnosed in a small herd of Holstein cattle. In this outbreak, 10 animals showed clinical signs of lead poisoning, 5 dead and others saved after chelating therapy with $CaNa_2EDTA$. Mean blood lead level of clinically intoxicated cattle was 0.624 ± 0.388ppm and the levels varied between 0.320 and 1.300 ppm. Blood and milk lead levels of 9 exposed but unaffected cattle ranged 0.250 to 0.590 and 0.060 to 0.290 ppm respectively. Although blood and milk lead levels of those cattle sharply decreased after removal of lead source, some of them showed higher than acceptable levels when samples analyzed 60 days later. Products of all lead exposed cattle may not be safe for human consumption for several weeks and analysis should be performed to ensure their safety.

Keywords: lead poisoning, cattle, battery recycling, Iran

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Introduction

Lead toxicity has been known since antiquity and lead poisoning is one of the most common environmental diseases affecting different animal species all over the world, as well as human beings. Old lead acid batteries of cars are the most common sources of lead poisoning in livestock. Lead based paints, used motor oils, automotive grease and oil filters, shotgun pellets, solder, leaded windows linoleum, smelter discharges and automobile exhaust are other lead sources for domestic animals (Cebra and Cebra, 2004; Radostits *et al*., 2007; Burren *et al*., 2010). These products can be ingested directly, pastures can be contaminated by them or prepared fees can be contaminated during storage or processing. Among domestic animals, cattle have been the most frequently affected due to its high susceptibility and natural curiosity, licking and indiscriminate eatinghabits (Waldner *et al*., 2002; Traverso *et al*., 2004; Sharpe and Livesey, 2006; Burren *et al*., 2010).

The absorption, distribution, storage and elimination of lead in animals depend on several factors including the chemical form of the lead, species, age and physiologic state of the animal, nutrition and rate of ingestion (Rumbeiha *et al*., 2001; Miranda *et al*., 2006; Pyne and Livesey, 2010). Most lead salts (such as lead sulphate) are more easily absorbed at a faster rate than metallic lead. Metallic lead from battery plates or other sources of metallic lead are retained in the forestomachs; slowly release lead and extending the period of exposure (Sharpe and Livesey, 2006).

Once absorbed from gastrointestinal tract, lead is initially distributed to soft tissues, kidneys and liver, by the blood. In the second phase, lead is redistributed from soft tissues to bone (Humphreys, 1991).The absorbed lead, some is excreted in the bile, urine and milk in lactating animals (Waldner *et al*., 2002; Radostits *et al*., 2007).

Case description

In December 2009 lead poisoning was diagnosed in a small dairy cattle herd of Holstein-bred in suburb of Mashhad. The farm had been reestablished by introducing of 25, 3-4 months pregnant cows. During past months, used batteries had been recycled in the vicinity of loose housing yards, in the place of fodder storage. Two weeks after entrance of cattle, signs of poisoning were observed and 4 cows were slaughtered within 7 days, because of lack of response to routine symptomatic treatments, lateral recumbency and poor prognosis. Other 6 cows were clinically examined. Clinical findings included frequent teeth grinding, salivation, inappetance, and ruminal atony, constipation with dark fetid feces, depression, head pressing and tachycardia. One of affected cows was sternally recumbent and showed dehydration, central blindness, hyperesthesia and trismus.

Blood samples were collected from theaffected cattle for lead analysis, hematology and serum biochemistry. Two samples of surface soil from fodder storage were also collected for lead analysis.Additional blood and milk samples were collected from 9 exposed but unaffected cows for lead analysis atthe day and 30 and 60 days after poisoning episode. At milk collection, precautions were taken to avoid any contamination from the teat skin and the environment.

Intoxicated cattle were treated by intravenous administration of calcium disodium EDTA (Merck-Germany) at the dose of 100mg/kg, intramuscular administration of thiamine hydrochloride (Nasr Co. Iran) at the dose of 20mg/kg andoral administration of magnesium sulfate at the dose of 500 mg/kg twice daily for 2 consecutive days. Clinical signs of poisoning were regressed by this therapeutic regimen and no additional mortality was occurred. No abortion was observed in exposed animals.

Whole heparinized blood and soil samples were processed for lead measurement using graphite furnace atomic absorption spectrophotometer (Perkin-Elmer 3030). For blood biochemistry, plasma fibrinogen andtotal protein, serum albumin, blood urea

nitrogen (BUN), creatinine, glucose, total billirubin, magnesium, calcium, phosphorous concentrations, and aspartate amino transferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (AP), lactate dehydrogenase (LDH) and creatine kinase (CK) activity were measured by Auto Analyser (Biotecnica, Targa 3000, Italy), using commercial kits.

Mean blood lead level of clinically intoxicated cattle was 0.624± 0.388ppm and the levels varied between 0.32 and 1.3ppm. The highest blood lead level belonged to a cow which showed nervous signs and recumbency. Total lead levels in 2 soil samples were 12575 and 15262ppm.

Blood and milk lead levels of exposed cattle in first sampling ranged 0.25 to 0.59 and 0.060to 0.290 ppm respectively. The ranges of blood and milk lead levels in second samples were 0.140 to 0.320 and 0.017 to 0.140 ppm respectively. Sixty days after elimination of lead source, those ranges were 0.130 to 0.230 ppm in blood and 0.007 to 0.082 ppm in milk. Time course of blood and milk lead levels is shown in Table 1.

Results of hematology and serum biochemistry are presented in Table 2. These results are showing high levels of serum CK, LDH, bilirubin and neutrophil counts. No basophilic stippling was observed in erythrocytes when blood smears stained by Giemsa. High CK and LDH activities were observed in one affected cattle associated with recumbency. All intoxicated cattle showed high serum activity of LDH.

Table 1. Time course of blood and milk lead levels (mean ± SD) of nonsymptomatic exposed cattle.

Sample	Day 1	Day 30	Day 60
Blood (ppm)	0.41 ± 0.11	0.22 ± 0.052	0.15 ± 0.063
Milk (ppm)	0.17 ± 0.094	0.065 ± 0.04	0.032 ± 0.027

Table 2. Levels of hematological and biochemical parameters in the lead intoxicated cattle.

Parameter	Affected cattle	Normal value
PCV%	33± 3.8	31.4 ± 3.2
WBC/ml	8570 ± 3749	7500 ± 1100
Neut/ml	3432 ± 1347	1800 ± 650
Eos/ml	190 ± 146	1200 ± 700
Lymph/ml	4697 ±	4000 ± 850
Mono/ml	328 ± 226	450 ± 200
Fibrinogen mg/dl	660 ± 151	300-700
Total protein g/dl	7.98 ± 0.43	7.6 ± 1.8
Albuming/dl	2.95 ± 0.31	3. 2 ± 1.3
Glucose mg/dl	65.5 ± 50.6	56.3 ± 14.1
Bilirubin mg/dl	0.68 ± 0.25	0.05 ± 0.12
Creatinine mg/dl	0.89 ± 0.12	1.5 ± 0.5
Urea mg/dl	26 ± 1.8	15 ± 5
AST u/L	116 ± 66.7	105.5 ± 46.9
ALP u/L	51 ± 21.1	194 ± 126
GGT u/L	16.6 ± 7.6	15.7 ± 4
LDH u/L	3166 ± 1145	1061 ± 222
CK u/L	2718 ± 4787	178.2 ± 101.2
Mg mg/dl	2.1 ± 0.6	2.45 ± 0.53
Ca mg/dl	8.5 ± 0.9	9.62 ± 0.85
Phosphorous mg/dl	5.7 ± 1.6	7 ± 1.55

Discussion

During an episode of lead poisoning in a group of cattle, as in this study, beside clinical cases, some animals show no symptoms but have elevated lead concentrations in blood and milk (Rumbeiha *et al*., 2001; Waldner *et al*., 2002; Sharpe and Livesey, 2006). Therefore, not only milk and offals of lead- poisoned cattle but also products of lead - exposed but non-affected cattle could be a source of lead for humans. Milk lead levels in some animals in this report were above than regulatory safety limits (0.050 ppm) even 60 days after discontinuing of lead exposure. Hence, animal products from such farms have to be controlled precisely regarding the public health aspects and prohibited their consumption when contain above residue limits.

Although lead contamination of animal products is probably insufficient to cause clinical lead poisoning in human beings, but low level of lead exposure can induced harmful consequences such asreduction in the intellectual and cognitive ability of young children (Meyer *et al*., 2003; Gilbert and Weiss, 2006).

The concentration of lead in the blood did not correlate well with the presence of clinical signs so that some asymptomatic animals showed high blood levels of lead as same as clinical cases, however clinical intoxicated cattle showed higher levels of blood lead levels. Speed of lead ingestion and individual characters may influence on blood lead levels.

The biological half life of blood lead in animals which accidentally exposed to lead varied tremendously between animals within and between herds.Blood lead half lives in between10.5 and 2507 days have been reported in lead intoxicated cattle (Rumbeiha *et al*., 2001; Waldner *et al*., 2002; Miranda *et al*., 2006; Radostits *et al*., 2007). However, it should be stressed that the half life of blood lead is difficult to evaluate in cases of accidental lead poisoning in cattle. The chemical and physical forms of lead affect the absorption. Metallic and sulfide forms are poorly absorbed, whereas acetate, phosphate, carbonate and hydroxide salts are assimilated more easily. Only a small amount, 1 – 2%, of ingested lead salts is absorbed (Cebra and cebra, 2004). Absorbed lead is rapidly distributed to soft tissues by the blood and deposited in the kidneys, liver and bone (Osweiler, 1996; Waldner *et al*., 2002). Lead is excreted through feces, urine and milk. Dietary factors, aging and physiologic statemay also affects the kinetics of lead inthe exposed animals (Osweiler, 1996).

On the other hand, the metallic lead tends to be retained in the forestomachs of ruminant animals. This provide a reservoir from which lead can continue to be absorbed and effectively extending the duration of exposure and half life of lead (Radostits *et al*., 2007).

In this study, exception of neutrophilia, no hematological changes were observed in the lead intoxicated cattle. Blood neutrophil elevation may be related to stress condition of the poisoning.

Although recycling of the lead acid batteries can be a factor to reduce pollution of the environment as well as reducing mining and extraction of new lead, lack of standard methods can be lead to contamination of the environment and animals and humans exposure. It is also concluded that lead exposed cattle even without clinical symptoms of poisoning, because retaining high blood and milk lead levels over a long period of time, are a risk for public health.

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مسمومیت با سرب در گاوهای شیری ناشی از بازیافت باتری، سطح بالای سرب شیر درگاوهای بدون نشانه‌ی درمانگاهی

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چکیده

مسمومیت با سرب ناشی از باقی مانده‌های بازیافت باتری در یک گله کوچک گاو هلشتاین تشخیص داده شد. در این مورد ۱۰ راس از دام‌ها نشانه‌های درمانگاهی مسمومیت را بروز داده و ۵ راس از آنها تلف شدند. به دنبال درمان اختصاصی با CaNa2EDTA تلفاتی در سایر دام‌های مبتلا رخ نداد. میزان متوسط سرب گاوهایی با نشانه‌های درمانگاهی ppm ۰/۶۲۴±۰/۳۸۸ و از ۰/۳۳ تا ppm ۱/۳ متفاوت بود. اندازه گیری سرب خون و شیر ۹ راس از گاوهای بدون نشانه درمانگاهی به ترتیب میزان ۰/۲۵ تا ۰/۵۹ و ۰/۰۶ تا ppm ۰/۲۹ را نشان داد. به دنبال حذف منبع سرب، این میزان‌ها با شیب تند کاهش نشان دادند ولی حتی بعد از ۶۰ روز نیز سطح سرب در برخی دام‌ها از مقدار قابل قبول بالاتر بود. گزارش حاضر نشان می‌دهد که فراورده‌های دامی گله گاوی که در معرض سرب قرار گرفته است تا هفته‌ها برای مصرف مناسب نبوده و با اندازه گیری سرب از سلامت آنها باید اطمینان حاصل شود.

واژگان کلیدی: مسمومیت با سرب، گاو، بازیافت باتری، ایران